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Welcome to DIALOG
Logon file001 15mar05 20:10:28


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Set      Items    Description
S1        5521    (STR OR GENOTYP?) AND (HEPATITIS OR HBV) AND INTERFERON
S2           0    S1 AND STR
S3           0    S1 AND STRP
S4          23    (STR OR STRP) AND INTERFERON
S5          17    RD (unique items)
S6          18    (SHORT(W)TANDEM(W)REPEAT?) AND INTERFERON
S7     4576709    6 NOT S4
S8           7    S6 NOT S4
? logoff h
       15mar05 20:28:29
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5/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013593314 BIOSIS NO.: 200200186825
Gleevec for relapsed CML after allogeneic transplant may overcome need for donor leukocyte infusions (DLI) or %interferon% (IFN)
AUTHOR: Rodriguez Roberto (Reprint); Snyder David (Reprint); Nademanee Auayporn (Reprint); O'Donnell Margaret (Reprint); Krishnan Amrita (Reprint); Bhatia Ravi (Reprint); Slovak Marilyn (Reprint); Resta Debra; Forman Stephen (Reprint)
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JOURNAL: Blood 98 (11 Part 1): p401a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Relapsed CML after allogeneic transplant (allo BMT) is usually treated with DLI or IFN. We explored whether Gleevec (formerly known as STI-571) could overcome the need for these therapies and their associated toxicity. Eight patients with relapsed (hematologic, n=7; cytogenetic, n=1) CML after allo BMT seen at City of Hope in the last year received treatment with Gleevec (n=7) or Gleevec+Ara-C (n=1) (7 of these patients were treated on Novartis protocols 001, 0113 or 0114). Median time from BMT to relapse was 12 months (mean 29.2 months; range 3-84 months). Disease status at relapse was chronic phase (CP) (n=4), accelerated phase (AP) (n=3), and blast crisis (BC) (n=1). Treatment for relapse prior to Gleevec included IFN (n=4) or DLI (n=1); all these patients failed to achieve hematologic remissions with these therapies. One patient in BC had a transient cytogenetic response after withdrawal of immunosupression. Median time from relapse to start of Gleevec was 6 months. Disease status at initiation of Gleevec was CP (n=3), AP (n=4) and BC (n=1). The starting dose of Gleevec was 400 mg/d for CP, and 600 mg/d for AP and BC. Active GVHD was present in 3 patients at the start of Gleevec. At a median follow-up of 4 months (mean 5.7 months, range 3-14 months), 4 patients (CP, n=2; AP, n=2) achieved and remain in complete cytogenetic (CCR) or molecular remission (MR) by PCR, 2 patients (CP, n=1; AP, n=1) are in complete hematologic response (cytogenetics and PCR not available), 1 patient in AP had a hematologic remission with persistent molecular disease but progressed after 3 months of therapy, and 1 patient in BC never achieved a hematologic response. All CCR patients tested for engraftment (n=3) reverted to >95% donor chimerism by %STR% or FISH analysis. Median time to CCR was 3 months (range 2-4 months). Responding patients have minimal or no GVHD, and only one remains on immunosuppression for recurrence of red cell aplasia (present before relapse). Gleevec was well tolerated; most patients developed transient cytopenias, but only 2 patients required dose modification of Gleevec because of rash and cytopenias, respectively. Conclusions: Gleevec can achieve CCR in patients with relapsed CML after allo BMT with minimal toxicity. If remissions prove durable, Gleevec may be preferred over DLI and IFN.

0009250558 BIOSIS NO.: 199497271843
Loss of heterozygosity and homozygous deletions on 9p21-22 in melanoma
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JOURNAL: Oncogene 9 (5): p1361-1365 1994 1994
ISSN: 0950-9232
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Recent studies have implicated chromosome 9p21-22 as a location for a gene involved in cutaneous melanoma (CM). Deletion mapping in 35 matched tumour-constitutional DNA pairs from metastatic melanomas (including one melanoma cell line) and one dysplastic naevus has been performed using six short tandem repeat polymorphic (%STRP%) markers (D9S157-D9S162-IFNA-D9S171-D9S126-D9S104) which span approximately 19 cM across the 9p21-22 region. Both heterozygous and homozygous deletions were observed across the region in melanomas from both sporadic and familial cases. Overall 57% (20/35) of the samples displayed some form of loss. A deletion map identifies two areas of common loss either side of the %interferon% gene cluster. Familial CM has previously been shown to link to the more proximal of these regions. The deleted region distal to IFNA has not been previously described in melanoma. The results imply the involvement of more than one tumour suppressor gene on 9p in CM.

0014665641 BIOSIS NO.: 200400036398
Simultaneous analysis of interleukin-10 gene microsatellites and single-nucleotide polymorphisms in parallel with tumour necrosis factor and %interferon%-gamma %short% %tandem% %repeats% by fluorescence-based polymerase chain reaction.
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JOURNAL: Genes and Immunity 4 (7): p459-468 October 2003 2003
MEDIUM: print
ISSN: 1466-4879 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Different cytokine genotypes exist in the population, for example, as a result of selective pressure of infectious diseases. It may be that specific cytokine genotypes that are beneficial by creating a 'proinflammatory' phenotype predispose to severe inflammatory disease with worse clinical outcome. There is individual variation in the production of certain cytokines in relation to their genotypes. IL-10, IFN-gamma and TNF-alpha are key components in the regulation of immune responses and the balance of their expression levels is predictive in certain diseases. To describe cytokine genotypes, a one-tube PCR reaction was developed to analyse simultaneously DNA sequence variations of cytokine genes IL-10, IFN-gamma, and TNF. This multiplex PCR approach was used to provide genotypic data for two geographically independent donor groups from Germany and Gabon. Significant differences were obtained for the majority of sequence variations comparing both populations. However, the SNPs within the 5'-flanking region of the IL-10 gene at position -1087 and -6208 are comparable in their genic and genotypic behaviour. Comparing allelic and genotypic disequilibrium between pairs of loci revealed different association patterns for both populations according to the geographical polymorphism. This assay may improve immunogenetic studies in disease, characterized by disbalanced IL-10, IFN-gamma and TNF-alpha expression.

0008736523 BIOSIS NO.: 199395038789
Assignment of a locus for familial melanoma, MLM, to chromosome 9p 13-p22
AUTHOR: Cannon-Albright Lisa A (Reprint); Goldgar David E; Meyer Laurence J ; Lewis Cathryn M; Anderson David E; Fountain Jane W; Hegi Monika E; Wiseman Roger W; Petty Elizabeth M
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JOURNAL: Science (Washington D C) 258 (5085): p1148-1152 1992
ISSN: 0036-8075
DOCUMENT TYPE: Article
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LANGUAGE: English

ABSTRACT: Linkage analysis of ten Utah kindreds and one Texas kindred with multiple cases of cutaneous malignant melanoma (CMM) provided evidence that a locus for familial melanoma susceptibility is in the chromosomal region 9p13-p22. The genetic markers analyzed reside in a candidate region on chromosome 9p21, previously implicated by the presence of homozygous deletions in melanoma tumors and by the presence of a germline deletion in an individual with eight independent melanomas. Multipoint linkage analysis was performed between the familial melanoma susceptibility locus (MLM) and two %short% %tandem% %repeat% markers, D9S126 and the %interferon%-alpha (IFNA) gene, which reside in the region of somatic loss in melanoma tumors. An analysis incorporating a partially penetrant dominant melanoma susceptibility locus places MLM near IFNA and D9S126 with a maximum location score of 12.71. Therefore, the region frequently deleted in melanoma tumors on 9p21 presumably contains a locus that plays a critical role in predisposition to familial melanoma.